

# Severe Lead Poisoning in the Plastics Industry: A Report of Three Cases

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**Background** Lead stabilizers (e.g., lead sulfate, lead stearate) are common additives in plastics used in electrical devices. In 1997, three plastics compounders at one California company were severely lead-poisoned.

**Methods** The poisonings were investigated by interviewing the workers, employer, and treating physician and reviewing medical records and environmental monitoring results. In addition to measuring blood lead levels (BLLs), noninvasive K X-ray fluorescence was used to measure bone lead concentration of the index case.

**Results** Blood lead concentrations of the three workers at time of diagnosis were 159, 114, and 108 µg/dl. The worker with highest exposure presented with clinical findings of crampy abdominal pain, constipation, normocytic anemia, fatigue, and reversible azotemia. Bone lead concentration in his tibia, calcaneous, and patella were 102, 219, and 182 ppm, respectively. The poisonings resulted from uncontrolled use of powdered lead sulfate stabilizer.

**Conclusion** Clinicians should be aware of potential serious overexposure to lead in compounding of plastics. Am. J. Ind. Med. 47:172–175, 2005. © 2005 Wiley-Liss, Inc.

**KEY WORDS:** lead; lead stabilizers; lead sulfate; plastics compounding; K X-ray fluorescence

## INTRODUCTION

Lead stabilizers such as lead sulfate or lead stearate are common additives in the formulation of polyvinyl chloride (PVC) plastic used to coat or insulate wire and cables. The lead acts as a stabilizer that protects the PVC from chemical degradation from heat and ultraviolet radiation. Lead pigments such as lead chromate also continue to be used to color plastics. When used in the manufacturing process, these lead

compounds are commonly supplied as powders, a form of the metal that may not be recognized as a hazard by workers or health care providers.

There is only limited blood lead level (BLL) data for the plastics industry in the United States in reports published in the last 15 years or available from state blood lead surveillance programs. In 1992, the Texas Health Department reported an investigation of lead poisoning at a manufacturer of plastic pigments. Seven of 22 workers had BLLs above 40 µg/dl (range: 43–107 µg/dl) [Centers for Disease Control, 1992]. Of the 35 states conducting blood lead surveillance, six provided BLL data for three plastics industries of interest (manufacture of plastics materials and resins, custom compounding of plastics, manufacture of current-carrying wiring devices<sup>1</sup>) to the authors. Since 1995, 144 individuals have been reported with BLLs at or above 25 µg/dl. Of these, 32 (22%) had BLLs at or above 40 µg/dl. This investigation documents the first identified cases of lead poisoning in the plastics industry in California.

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## CASES

In California, clinical laboratories are required to report all BLLs to the California Department of Health Services (CDHS). On November 12, 1997, the Occupational Lead Poisoning Prevention Program (OLPPP) in CDHS received two reports of an adult with a severely elevated BLL (159 and 164  $\mu\text{g}/\text{dl}$ <sup>2</sup>, performed 1 week apart). As part of its standard protocol to respond immediately to all BLLs  $\geq 60 \mu\text{g}/\text{dl}$ , OLPPP interviewed the affected individual (a compounder for a plastics manufacturer), his employer, and the treating physician within a day of receipt of the BLL reports. The initial interviews revealed that the worker had symptomatic lead poisoning resulting from uncontrolled use of a powdered lead sulfate stabilizer in custom compounding PVC plastic pellets intended for use in electrical devices. Although the index case had used this lead sulfate product at his current workplace for approximately 5 years, the employer had never provided any lead-related industrial hygiene monitoring or BLL testing.

At OLPPP's recommendation, the employer provided BLL testing for the index case's eight co-workers in the company's production department. The two other compounders had markedly elevated BLLs (114 and 108  $\mu\text{g}/\text{dl}$ , respectively) and were symptomatic. All three lead-poisoned workers were removed immediately from lead exposure and chelated by a board-certified occupational medicine physician. The six remaining workers had BLLs ranging from 4 to 48  $\mu\text{g}/\text{dl}$ . For comparison, the geometric mean BLL for the United States general population and working aged adults is less than 2  $\mu\text{g}/\text{dl}$  [Centers for Disease Control and Prevention, 2003]. Follow-up BLL testing of workers' children also identified one child, a 3-month-old infant, with an elevated BLL (10  $\mu\text{g}/\text{dl}$ ) [Centers for Disease Control, 1991].

The three poisoned workers were questioned about potential sources of lead exposure outside of their employment at the plastics company. None were engaged in other jobs or hobbies associated with lead exposure. The index case had been employed fulltime at the plastics compounder since mid-1992. Between 1988 and 1991 he had performed plastics compounding for another employer where powdered ingredients were rarely used and where he doubted that lead additives had been encountered. Between 1985 and 1987 he had been employed doing plastic extrusion molding but did not do compounding. In the first part of 1992, the index case was employed as a compounder of rubber resins.

The review of the index case's medical history indicated that he reported chronic recurrent abdominal pain and muscle "spasms" in his extremities to his personal physician

beginning in approximately February 1996. Between January and November 1997, he suffered three severe bouts of abdominal pain accompanied by fatigue, decreased appetite, marked constipation, and the inability to find a comfortable position while resting in bed at night. The first two episodes, which gradually increased over a 3-day period and were exacerbated by movement, subsequently abated over the course of 1 week. Medical consultation produced a presumptive diagnosis of gastritis or peptic ulcer, and  $\text{H}_2$  antagonists were prescribed; calcium supplementation was recommended for the muscle spasms. At the time of the third episode, in November 1997, the worker requested a BLL because he had noticed that the bags of powder he worked with had "lead" printed on them and were marked with a skull and crossbones. In addition to the BLL of 164  $\mu\text{g}/\text{dl}$ , laboratory evaluation at that time revealed a serum creatinine of 2.9 mg/dl (normal range: 0.7–1.3), blood urea nitrogen 34 mg/dl (7–18), hemoglobin 11.0 g/dl (13.5–17.5), hematocrit 33.6% (40–54), and MCV 87 c/mcu (80–94).

Following the worker's removal from the workplace and the first week of chelation with oral succimer, further evaluation of his renal function revealed a creatinine clearance of 49 ml/min (normal range: 75–125); urinalysis and abdominal ultrasound were negative. Two weeks later, the serum creatinine had fallen to 1.4 mg/dl and remained between 1.2 and 1.4 mg/dl on multiple follow-up measurements over the following 8 months. BLL at the end of chelation reached a nadir of 19  $\mu\text{g}/\text{dl}$ , but rebounded four weeks later to 54  $\mu\text{g}/\text{dl}$ . Measurement of bone lead concentration by noninvasive, K X-ray fluorescence in October 1998 revealed a tibial (cortical bone) lead concentration of  $102.1 \pm 3.0$  ppm, calcaneus (trabecular bone) lead  $219.1 \pm 7.1$ , and patellar (trabecular bone) lead  $181.6 \pm 7.0$  (all results approximately 10-fold greater than predicted for a male of his age). BLL at that time was 45.4  $\mu\text{g}/\text{dl}$ .

## Investigation and Follow-Up

This small employer custom compounds vinyl pellets from raw ingredients (powdered PVC resin, powdered stabilizer, and liquid lubricant). Lead sulfate was added as a stabilizer when formulating plastics for manufacturers of products such as insulated electrical wiring. Compounders frequently (often daily) mixed batches that used pure lead sulfate as the stabilizer. Fifty-pound bags of lead sulfate were cut open and left unsealed in the production area; for each batch, a compounder hand-scooped six to eight pounds of pure lead sulfate powder and dumped it into a large mixing vat while wearing a paper dust mask. Up to 34 batches were mixed per day. The intermediate product, a fine compounded powder, came out a chute into an open bin at the bottom of the mixer. When the bin was full, it was moved by forklift to the other side of the plant where it was dumped into an extruder to form solid pieces of plastic that were then fed into a

<sup>2</sup> The Occupational Safety and Health Administration requires medical removal of a worker with a blood lead level  $\geq 60 \mu\text{g}/\text{dl}$  or an average BLL  $\geq 50 \mu\text{g}/\text{dl}$  on the last three tests, or all tests over the previous 6 months.

pellitizer to make the final product—pellets approximately 1/2" long and 1/4" in diameter.

There was no lead safety program at the company, and air monitoring had never been performed. There was a local exhaust ventilation system at the mixing station, but it was not evaluated regularly. Cleanup was done by dry sweeping the shop floor. Workers ate and smoked in the production area, and some wore their work clothes and shoes home. The index case reported using a compressed air hose to blow dust off his work clothes prior to wearing them home. There were no showers at the worksite.

The employer was unaware that lead sulfate posed a serious health hazard and of his responsibilities under the OSHA General Industry Lead Standard (29 CFR 1910.1025). He had relied on the product Material Safety Data Sheet (MSDS) for information on health effects and guidance on protecting his workers.<sup>3</sup> The MSDS for this product did not mention the effects of lead on the central nervous, blood-forming, and reproductive systems, or its ability to cause kidney damage. The MSDS recommended that a "respirator for fine dust" be used; the employer mistakenly thought this meant a dust mask, since there was no mention of HEPA (high efficiency particulate) filters, which he had seen specifically recommended on MSDSs for other products. Had the MSDS recommended the appropriate respirator, a half-face respirator with HEPA filters, the employer stated that he would not have relied on dust masks to protect his employees.

OLPPP made specific recommendations to correct problems identified during employer and worker interviews including significant changes in work practices, implementation of a respirator program, establishing a medical surveillance program, conducting personal air monitoring for lead, and training workers.

Air monitoring, done several weeks after the index case was identified, showed personal air lead levels of 460–1,100  $\mu\text{g}/\text{m}^3$  (time-weighted average for sampling times of 2.8 to 3.9 hr; the OSHA permissible exposure limit is 50  $\mu\text{g}/\text{m}^3$ , 8 hr time-weighted average). In the year after the index case was identified, the company switched to a pellet formulation of the stabilizer to reduce lead dust. Follow-up personal air monitoring done in December 1998 continued to show excessive air lead levels (20–400  $\mu\text{g}/\text{m}^3$ , 8 hr time-weighted average). In a further effort to reduce exposure, the company switched to a pre-packaged stabilizer in a dissolvable package that was added intact to the mixer. During this time, compounds were required to wear half-face respirators with HEPA (P-100) filters. Air monitoring conducted during use of the pre-packaged stabilizer still showed excessive lead levels. Personal air lead levels ranged from

3 to 210  $\mu\text{g}/\text{m}^3$  (8 hr time-weighted average). The source of the continued high air lead levels was not clear. At the time of monitoring, white powder was visible on the floor and surfaces throughout the shop and was likely kicked up into the air by dry sweeping, etc. The powder may have been lead sulfate stabilizer left over from the time before the switch to the pre-packaged stabilizer, or may have been dust from the intermediate product, a fine compounded powder.

Because of continuing difficulty controlling lead exposures, even with the use of a pre-packaged stabilizer, as well as a desire to go lead-free, the company began to research lead-free substitutes. After a lengthy testing process, the company found a lead-free plastic formulation that met Underwriters Laboratories, Inc.<sup>®4</sup> performance requirements and was satisfactory to their customers. In November 2000 the company switched to the lead-free stabilizer.

In 1998, the Society of the Plastics Industry, at OLPPP's request, alerted their membership to this potential problem via their newsletter and posted an alert on their website. OLPPP also sent a letter in February 1999 to the 392 California employers that compound plastic resins, or manufacture plastics, resins, or current-carrying wiring devices.

OLPPP notified Cal/OSHA of the deficient MSDS in April 1998. OLPPP also notified federal OSHA in April 1999, and OSHA contacted the stabilizer importer in December 1999 to correct the MSDS.

## DISCUSSION

### Index Case

At the time of his initial blood lead measurement of 159  $\mu\text{g}/\text{dl}$ , the index case had exhibited a constellation of signs and symptoms characteristic of overt chronic lead poisoning, including episodic bouts of crampy abdominal pain, constipation, fatigue, and normocytic anemia [Lilis et al., 1968; Hamilton and Hardy, 1974; Pagliuca et al., 1990; Kosnett, 2005]. The reversible renal insufficiency exhibited by the patient is a less common feature of severe lead poisoning. At the time of his presentation in November 1997, the patient had a serum creatinine that peaked at 2.9  $\text{mg}/\text{dl}$ , and a creatinine clearance of 49  $\text{ml}/\text{min}$  (normal: 75–125). The patient's serum creatinine declined during lead chelation with succimer, and by the spring of 1998 had appeared to stabilize in the range of 1.2–1.4  $\text{mg}/\text{dl}$ . The reversibility of renal insufficiency occurring as a result of massive acute lead exposure (or acute on chronic exposure), has been described in the medical literature [Radosevic et al., 1961; Lilis et al., 1968]. Rather than reflecting tubular interstitial scarring, a sequelae of chronic lead exposure that is irreversible, the

<sup>3</sup> The MSDS lists the hazardous ingredients in a product, describes its health and safety hazards, and recommends appropriate measures for controlling exposure. The manufacturer of a product containing hazardous substances is required by law to develop an MSDS. Employers are required by law to make this MSDS available to employees who use the product (29 CFR 1910.1200).

<sup>4</sup> Underwriters Laboratories, Inc.<sup>®</sup> is an independent, not-for-profit product safety testing and certification organization.

reversible component may be due to a vasoconstrictive effect of high lead levels on renovascular smooth muscle [Lilis et al., 1968; Chai and Webb, 1988], possibly akin to the reversible increases in intestinal smooth muscle tone associated with lead colic [Janin et al., 1985]. Improvement in lead-induced renal insufficiency following succimer chelation has been described in an animal model [Khalil-Manesh et al., 1992]. Although the patient's serum creatinine declined from the peak value of 2.9, it remained significantly higher than his value of 1.0 measured in May 1996. Thus, a component of subclinical chronic lead nephropathy may also be present.

Because greater than 90% of the body lead burden resides in bone, where it persists with a half-life of years to decades, non-invasive measurement of lead in bone by K X-ray fluorescence has found clinical utility as a biomarker of long-term, cumulative lead exposure. In several workplace studies, a single measurement of lead in bone has correlated strongly with time-integrated blood lead measurements accumulated from years of past biological monitoring [Roels et al., 1995]. Although no prior BLLs were available for the index case, his markedly elevated bone lead concentrations, at the upper range of bone lead values found in long-term lead smelter workers [Gerhardsson et al., 1993; Fleming et al., 1998], are consistent with his having sustained years of high dose lead exposure as a plastics compounder and rubber maker.

## Prevention Strategies/Recommendations

Employers commonly rely on MSDSs for determining hazards and appropriate worker protections since manufacturers must by law supply them and they are therefore readily available. However, the quality of MSDSs varies greatly and can be grossly inadequate. Neither federal OSHA nor Cal/OSHA routinely reviews MSDSs for accuracy and completeness. This incident points out the danger of relying solely on MSDSs for accurate health effects and worker protection information. Employers should always be encouraged to seek additional health and safety information on the hazardous materials in the products they use from OSHA consultation programs, state health department occupational health programs, workers' compensation carriers, or private occupational health and industrial hygiene consultants. Trade associations can also assist the hazard communication process by informing member companies of potential hazards associated with materials or products commonly used in relevant industrial processes.

This example demonstrates that even engineering controls, such as switching to a pre-packaged stabilizer, may not be sufficient to control lead exposures. In this instance air

lead levels continued to be excessive even with the use of the pre-packaged product. Substitution of lead-containing stabilizers with a less hazardous lead-free stabilizer is the surest way to protect workers.

The employer in this case was a small business manufacturing a specialty product. There are likely other companies who perform similar work. Occupational medicine providers, industrial hygienists, and OSHA inspectors should be aware of the possibility of significant lead exposure in the plastics industries.

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